# Cardiology is improving, focusing on INFLAMMATION, not just cholesterol, to stop heart disease

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For generations, the battle against heart disease has been fought with a single, glaring spotlight fixed on cholesterol. Patients left clinics with statin prescriptions, their health distilled to a number on a lipid panel, believing that controlling this one metric was their shield against heart attack and stroke. This cholesterol-centric model, however, may have missed the true underlying issues behind cardiovascular disease.

A paradigm-shifting directive from the American College of Cardiology (ACC) is now redirecting the medical community's gaze to a more pervasive and fundamental culprit: chronic inflammation. The newly released 2025 ACC scientific statement does not merely suggest an add-on test; it calls for a fundamental rethinking of cardiovascular prevention, placing the measurement and management of inflammation on equal, if not superior, footing to cholesterol management. This move, decades in the making, validates a growing body of science suggesting that the real engine driving arterial plaque from a benign buildup to a life-threatening rupture is not passive lipid accumulation but an active, inflammatory war within our blood vessels.

**Key points:**

- The American College of Cardiology now recommends universal screening for chronic inflammation using the high-sensitivity C-reactive protein (hsCRP) test for both primary and secondary cardiovascular disease prevention.

- Decades of research show that inflammation is a stronger predictor of future heart attacks and strokes than LDL cholesterol, even in patients who have achieved very low cholesterol levels with statin therapy.

- This "residual inflammatory risk" explains why many patients on optimal drug therapy still suffer cardiovascular events, pointing to a previously untreated component of the disease.

- The new guidelines endorse proven anti-inflammatory lifestyle interventions, like the Mediterranean diet and regular exercise, and recognize the role of specific anti-inflammatory medications like low-dose colchicine for high-risk patients.

- This shift represents a move from a simplistic "plumbing" model of heart disease to a complex biological understanding where inflammation damages the arterial wall, making it susceptible to cholesterol deposition and eventual catastrophic plaque rupture.

## The evidence that rewrites the textbook

The ACC's bold stance is not built on a hunch but on a mountain of evidence accumulated over 30 years, much of which lay in plain sight while clinical practice remained fixated on

lipids. One landmark study followed nearly 28,000 initially healthy women for three decades. The findings were stark: a single measurement of hsCRP, a marker of inflammation, provided a greater spread of risk for future cardiovascular events over those 30 years than a measurement of LDL cholesterol. The inflammation signal was simply more telling.

Perhaps more damning for the old paradigm is data from patients already undergoing intensive treatment. Analysis of over 31,000 patients in major contemporary trials who were already taking statins revealed that elevated hsCRP was a stronger predictor of subsequent heart attack, stroke, or cardiovascular death than their LDL cholesterol level. This phenomenon is termed "residual inflammatory risk." Imagine a patient who has dutifully taken their medication and achieved an LDL level of 70 mg/dL—a number most cardiologists would celebrate. If that patient's hsCRP remains above 2 mg/L, their risk of a major event remains alarmingly high. A Swedish registry of over 84,000 atherosclerosis patients found that 60% had elevated hsCRP despite modern care, and they were the ones experiencing poor outcomes. For these individuals, the fire was still burning, even if the grease trap had been cleaned.

The biological rationale clarifies why this correlation is causal. Atherosclerosis is not a bland pipe-clogging process. **It is an inflammatory disease.** Chronic, low-grade inflammation, often stemming from factors like poor diet, smoking, stress, or excess visceral fat, creates microscopic injuries in the delicate endothelial lining of arteries. This injury triggers an immune response. White blood cells rush to the site, infiltrate the arterial wall, and become engorged with cholesterol, forming "foam cells" and the earliest fatty streaks. The plaque that develops is not inert; it is a metabolically active, inflamed tissue. This inflammation weakens the plaque's fibrous cap, making it vulnerable to rupture. When it ruptures, the body's clotting mechanisms spring into action, often creating a thrombus that fully blocks the artery, leading to a heart attack or stroke. In this sequence, cholesterol is a participant, but inflammation is the instigator and director.

## From measurement to management: Taming the internal flame

The ACC statement's core clinical recommendation is straightforward: "Clinicians will not treat what they do not measure." It advocates for universal hsCRP screening in stable patients. A level below 1 mg/L indicates lower risk, 1 to 3 mg/L indicates average risk, and above 3 mg/L indicates higher cardiovascular risk, independent of cholesterol numbers.

Management then follows a two-pronged approach: lifestyle as foundation and pharmacotherapy for those at highest risk. The guidelines strongly emphasize anti-inflammatory lifestyles, pointing to the profound success of the PREDIMED trial, where a **Mediterranean diet supplemented with extra-virgin olive oil or nuts reduced major cardiovascular events by 30%** compared to a low-fat diet. This diet, rich in polyphenols from plants, omega-3 fatty acids from fatty fish, and monounsaturated fats from olive oil, actively dampens inflammatory pathways. The science of specialized pro-resolving mediators

(SPMs)—molecules derived from omega-3s that actively signal the body to resolve inflammation—provides a mechanistic explanation for these dietary benefits. Regular physical activity and smoking cessation are also powerfully anti-inflammatory.

For patients with established disease and persistent high hsCRP despite statins, the guidelines point to low-dose colchicine. This ancient, inexpensive drug has shown in trials like COLCOT and LoDoCo2 to reduce recurrent cardiovascular events by approximately 25%. It represents the first FDA-approved anti-inflammatory drug specifically for atherosclerotic cardiovascular disease. The statement also notes ongoing research into more targeted biologic agents, such as interleukin-6 inhibitors, for high-risk groups.

For decades, a chorus of researchers and physicians questioned the primacy of the cholesterol hypothesis. Autopsy studies as far back as the 1930s, and as recent as the early 2000s, frequently found poor correlation between serum cholesterol levels at death and the severity of atherosclerosis in the arteries. Trials like the Lyon Diet Heart Study in the 1990s, which showed a 70% reduction in mortality with a Mediterranean diet without major cholesterol lowering, stood in stark contrast to the concurrent statin trials. Critics pointed out that statins themselves have pleiotropic, or multiple, effects, including anti-inflammatory properties, which may be responsible for a significant portion of their benefit, a notion supported by the fact that their clinical benefit often correlates better with reductions in hsCRP than with reductions in LDL.

The new ACC guidelines, in many ways, bridge this historical divide. They do not discard cholesterol management; they contextualize it within a broader inflammatory framework. They affirm that for a large subset of patients—those with "residual inflammatory risk"—lowering cholesterol alone is insufficient.

This more nuanced model offers a powerful explanation for individual variability in heart disease risk and provides a new set of tools, both simple and sophisticated, to protect the human heart. It moves cardiology from a mechanic's approach to that of a biologist, addressing the root cause of the damage, not just its most visible symptom.

**Sources include:**

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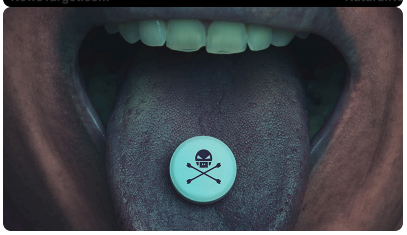

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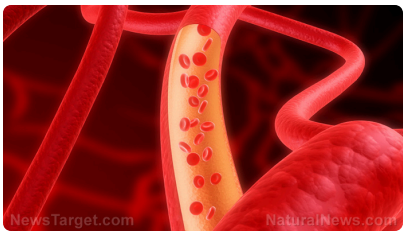

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